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Investigation of histopathological alteration of paramyxovirus-1 in naturally infected racing pigeons

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Abstract

The current study aimed to investigate the infection of racing pigeons with paramyxovirus-1(PMV-1) based on clinical signs of histological and histochemical changes, which increase the mortality rate in flocks called pigeons paramyxovirus. The study samples were collected from 45 birds from different Mosul city regions suffering from neurological signs considered pathological signs of paramyxovirus infection, like tremors, ataxia, leg and wing paralysis, and torticollis. The study collected some organs from birds most affected by infection with paramyxovirus. They include the heart, brain, liver, and kidneys. Grossly, brain lesions represented 66.6%, heart lesions represented 37.7%, liver lesions represented 55.5%, and kidney lesions represented 42.2%, respectively. The histological examination of these organs showed many histological changes represented by degenerative, necrotic, and inflammatory changes in all the tissue studied, in addition to the presence of vascular changes and fibrosis in some sections. In contrast, the section stained with Masson's trichrome showed severe deposition of collagen fibers in liver and heart tissues. The conclusion of the current study represented the histopathological histochemical changes in naturally infected racing pigeons with PMV -1. pigeons are considered a serious victim of the virus, which replicates in all body organs, since it is considered a severe threat and problem to poultry flocks.

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Introduction

Newcastle disease (ND), one of the most contagious ailments affecting poultry, is classified by the World Organization for Animal Health as having significant importance on international trade and affecting about 240 species of birds, including pigeons (1-4). ND is among the most critical two bird diseases (fowl pox and avian influenza) because of high mortality and economic importance (5-9). New castle virus (NDV), called avian paramyxovirus type 1 (APMV-1), was firstly isolated from chicken but also isolated from other species like turkeys, pheasants, game birds, pigeons, and ostriches (10-12). ND in pigeons (*Columba livia*) is called pigeon paramyxovirus type 1(pPMV-1) and is caused by genotype VI Newcastle disease virus, which is closely related to ND in poultry; firstly, observed in 1980 in the Middle East and then spread rapidly around the world and became endemic in wild and domestic pigeons in many countries (13-15). Infection with pPMV-1 in pigeons occur at all age and in any season. Pigeons are considered natural reservoirs for PPMV-1. The clinical signs in infected pigeons depend on the virulence of the specific isolate and the host's immunity (13-17). This virus replicated in different tissues, mainly the brain and lung, kidney, trachea, spleen, liver, and bursa of Fabricius and pancreas; this virus causes high morbidity and mortality in pigeons (18-20). pPMV1 causes outbreaks among the flock and threatens chickens and other birds. Pigeons suffer from neurological signs of profuse greenish and hemorrhagic diarrhea. The serious problem of this virus is the alteration in the pPMV-1 genome composition and the transmission to chickens (21-23). nervous signs include neck tremors,

torticollis, unilateral paralysis, and unbalanced gait. Other symptoms include weight loss, depression, and ruffled feathers; some pigeons show diarrhea without nervous signs (24). Polydipsia, polyuria, and torticollis without weight loss are the main signs of pPMV-1 infection. This distinguishes it from other diseases with the same signs (25). PPMV-1 has become most prevalent in recent years in pigeons, and asymptomatic pigeons have a significant role in infection (13). Treating sick birds may decrease the severity of infection (17).

Because of the few reports about the histopathology of pPMV1 in pigeons, this study aimed to detect the infection with NDV1 in pigeons in Mosul city by the histochemical and histopathological view and determine the clinical signs science histological lesions is significant in the pPMV-1 infection diagnosis.

Materials and methods

Ethical approve

The College of Veterinary Medicine at the University of Mosul in Iraq conducted this work under the ethical approval number UM.VET. 2022.033 from the IACUC.

Source of pigeons

The diseased pigeons(*Columbia livia*) showed nervous signs, torticollis, ataxia, ruffled feather, greenish diarrhea, and respiratory signs collected from different areas in Mosul city, veterinary medical clinics in addition to case received by Teaching Hospital at University of Mosul from December 2022 to February 2023, number of pigeons reached to 45 bird weighted 150-200 gm which submitted to the College of Veterinary Medicine at the University of Mosul's department of veterinary pathology and poultry disease.

Sample collection

After clinical examination of the diseased birds, the Clinical signs were reported, the diseased pigeons were Euthanized, and postmortem findings were reported, samples collected like the brain kidneys; for histological and histochemical diagnosis, the liver and heart were marked and then fixed in 10% formalin for 72 hours.

Histopathology

Samples were embedded in paraffin, using routine tissue processing, sectioned at 4-5 µm, and stained with Hematoxylin and eosin (H&E) for histopathological examination (26). Sections were examined by using an Olympus light microscope at magnification power 10 and 40x and imaged using a microscope digital camera.

Histochemical staining

Samples were embedded in paraffin, using routine tissue processing, sectioned at 4-5 µm, stained with Masson Trichrome Stian (26). section was examined by using an Olympus light microscope at magnification power 10x and 40x and imaged using digital camera.

Results

Clinical signs

Surveys showed that the beginning of ND in pigeons occurred suddenly. The diseased pigeons showed in-appetence and nervous signs like tremors, ataxia, leg and wing paralysis, torticollis, greenish diarrhea, and ruffled conjunctivitis. periocular edema, dyspnea, sneezing, tremors and dehydration (Figure 1).

Figure 1: A: pigeons (*Columbia livia*) show leg paralysis, B: pigeon (*Columbia livia*) show neck torticollis. C: greenish diarrhea from affected pigeon (*Columbia livia*).

Gross pathological changes

Postmortem changes revealed congestion of meningeal blood vessels of the brain, kidneys were congested and enlarged, and white disseminated pinpoint foci with hemorrhage on livers showed multiple necrotic foci with hypertrophied congested heart (Figure 2). The number of affected organs is 30, 17, 25 and 19 in brain, heart, liver and kidney respectively with brain in percentage of 66.6%, heart with percentage 37.7%, liver is 55.5% while kidney is 42.2%. (Table 1).

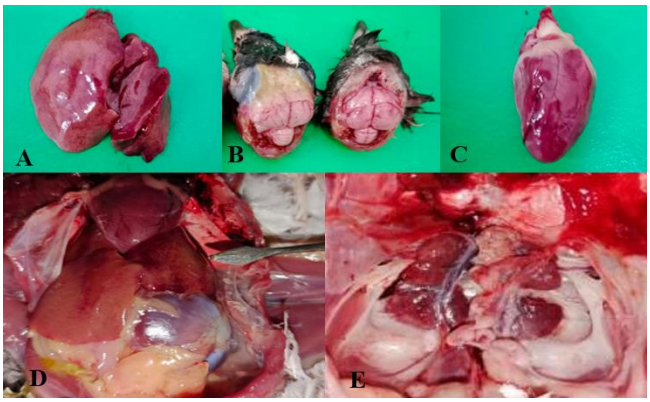

Figure 2: A: The liver of a pigeon (*Columbia livia*) shows white foci of necrosis. B: The brains of pigeons (*Columbia livia*) show severe congestion of meningeal blood vessels. C: heart enlarged with a white area representing myocardial necrosis, D: white foci of necrosis on the liver, E: kidney of pigeons (*Columbia livia*) shows congested with foci of necrosis.

Table 1: Gross pathological lesions of pigeon

Organ	Numbers	Percentage
Brain	30	66.6%
Heart	17	37.7%
Liver	25	55.5%
Kidney	19	42.2%

Histopathological findings

Pigeons with p PMV-1 infection were examined histopathologically, and congestion was seen in meningeal capillaries and hemorrhage in brain tissue in addition to vacuolar degeneration, vasogenic edema, and gliosis. Lymphohistiocytic infiltration with perivascular cuffing of lymphocyte, demyelination, and encephalomalacia swelling of Purkinje cells with cystic formation. (Figures 3-6). The liver section showed focal infiltrations of inflammatory cells with deposition of fibrin around the necrotic hepatic tissue (Figure 7), some sections showed necrotic foci infiltrated with inflammatory cells, deposition of fibrin around the necrotic cells, dilatation of sinusoids, multifocal areas of hepatic necrosis, congestion of central vein, other sections showed fibrosis around central vein with hyalinization of the blood vessel wall (Figures 8 and 9) and section that stains with Masson's trichrome shows sever deposition of collagen fibers (Figures 10 and 11). The kidney section showed interstitial nephritis, degeneration, and necrosis of renal epithelium with lymphoplasmacytic infiltration with hyaline cast and cystic formation. The other section showed cloudy swelling of epithelial cells (Figures 12 and 13). Heart sections showed edema and vacuolated cardiac muscle fibers with infiltration of lymphocyte and plasma cells. myocardial sections revealed necrotic cardiomyocytes and vacuolation in cardiac myofiber with loss of striation of cardiac myocytes and cardiomyopathy with hyalinization of blood vessels and deposition of collagen fibers in some sections stained with Masson's trichrome (Figures 14-18).

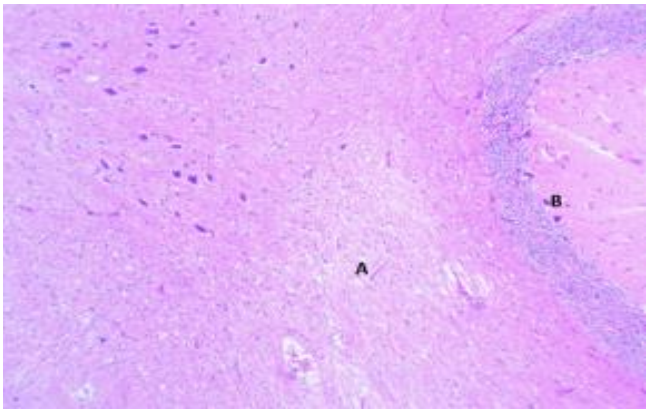

Figure 3: Histological section of brain tissue of racing pigeon. (A) Demyelination and encephalomalacia, and (B) degeneration and necrosis of Purkinje cell. H&E. 100X.

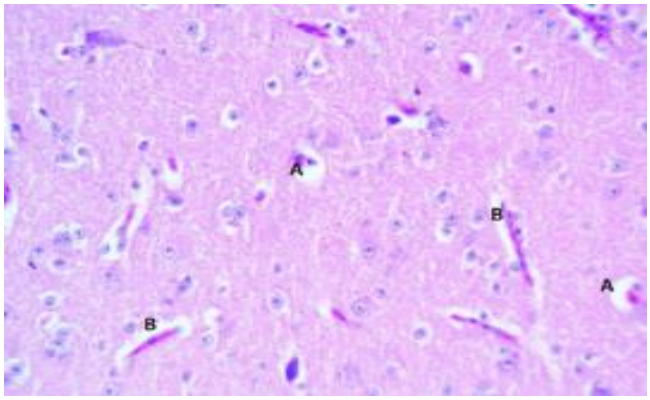

Figure 4: Histological section of brain tissue of racing pigeon. (A) Neuronal degeneration and vacuolation of the neuron, and (B) vasogenic edema. H&E. 400X.

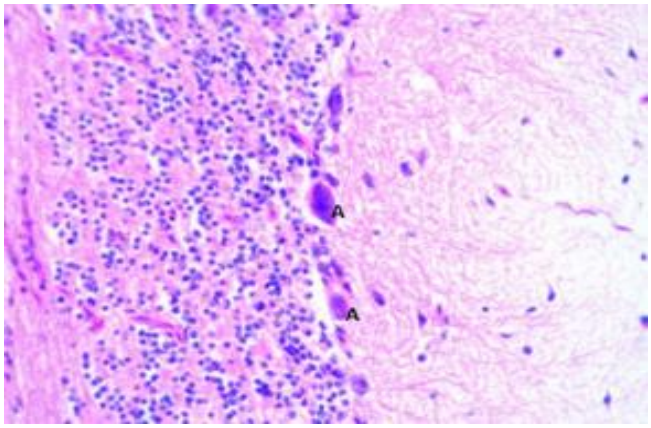

Figure 5: Histological section of brain tissue of racing pigeon. (A) Swelling of Purkinje cell and distortion from its position. H&E. 100X.

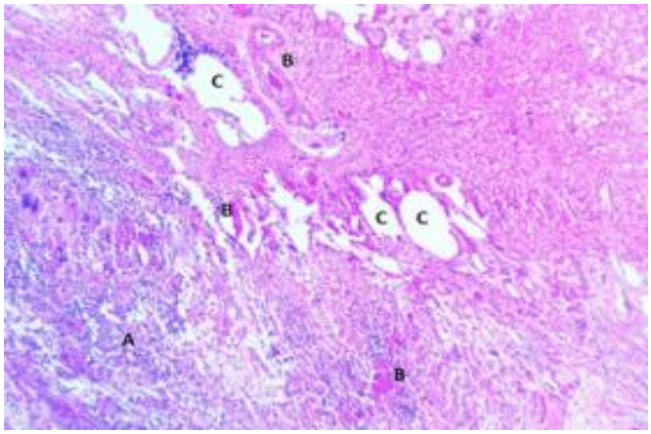

Figure 6: Histological section of brain tissue of racing pigeon. (A) Massive lymphoplasmacytic infiltration, (B) congestion of meningeal blood vessel, and (C) cystic formation in brain tissue. H&E. 100X.

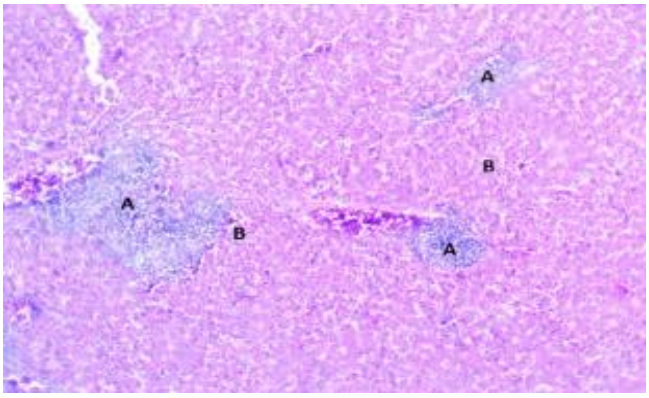

Figure 7: Histological section of the liver of a racing pigeon. (A) Focal infiltration of inflammatory cells, and (B) deposition of fibrin around the necrotic tissue. H&E. 100X.

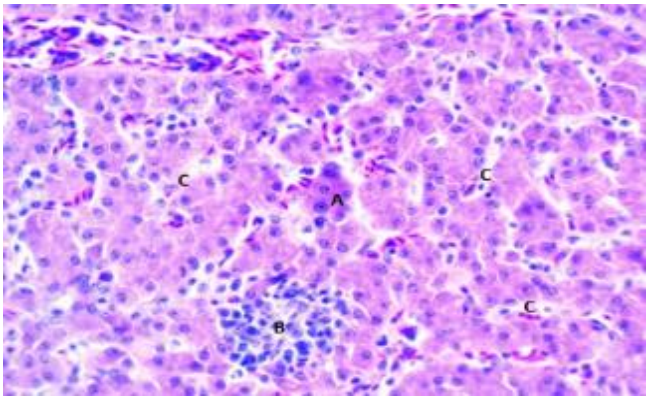

Figure 8: Histological section in racing pigeon of the liver. (A) Nucleus of hepatocyte undergoes pyknosis and Karrolysis, (B) foci of inflammatory cell, and (C) dilatation of sinusoids. H&E. 100X.

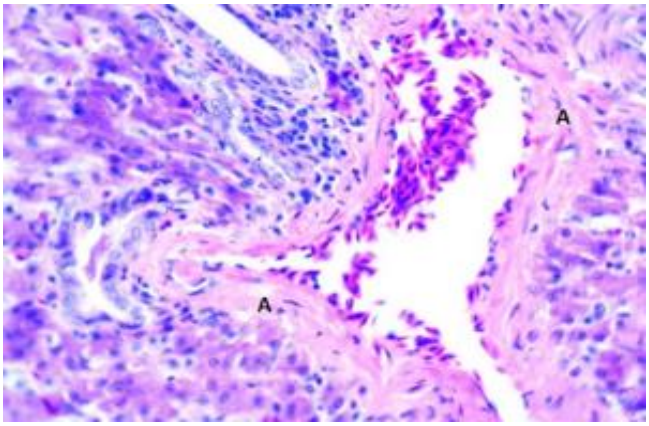

Figure 9: Histological section in the liver of racing pigeon. (A) Fibrosis around the blood vessel, and (B) lymphoplasmacytic infiltration around the blood vessels. H&E. 400X.

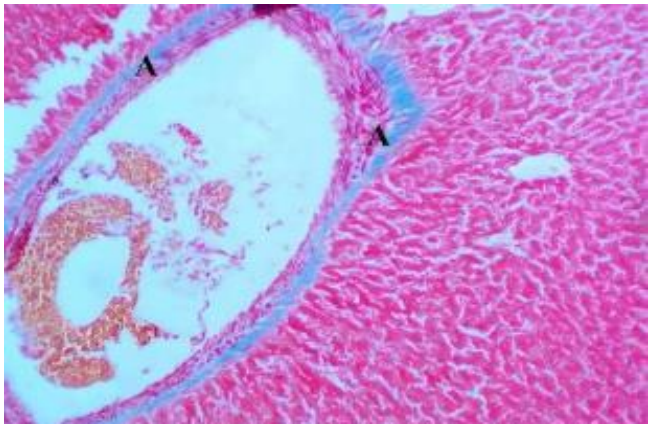

Figure 10: Histological section in the liver of racing pigeon. (A) Fibrosis around the blood vessel takes the blue color of the stain. Masson's trichrome stain.100X.

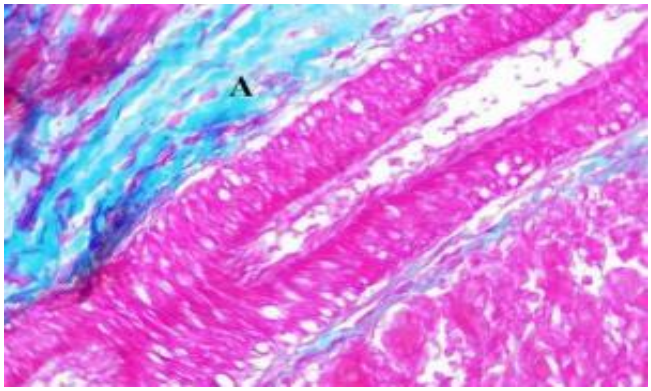

Figure 11: Histological section in the liver of racing pigeon. (A) Deposition of collagen fibers between hepatocytes takes the blue color of the stain. Masson's trichrome stain.100X.

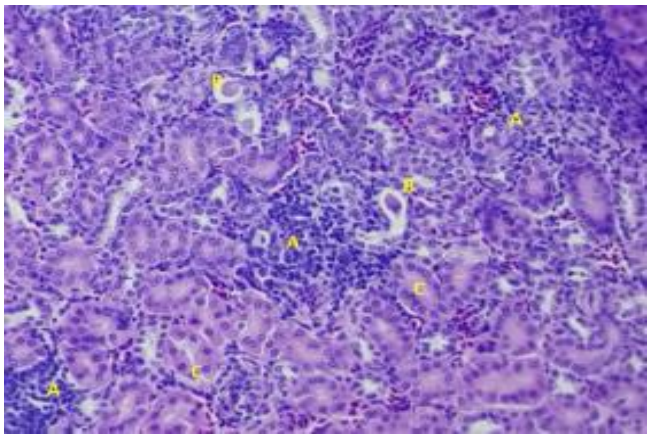

Figure 12: Histological section in the kidney of racing pigeon. (A) Infiltration of lymphocytes and plasma cells, (B) cystic formation, and (C) degeneration and necrosis of the renal tubules. H&E. 400X.

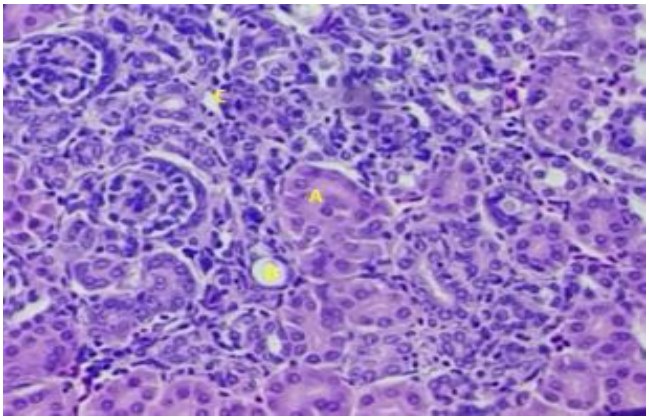

Figure 13: Histological section in the kidney of racing pigeon. (A) Degeneration and necrosis of the renal epithelium, (B) cystic formation, and (C) infiltration of lymphocyte and plasma cells. H&E. 400X.

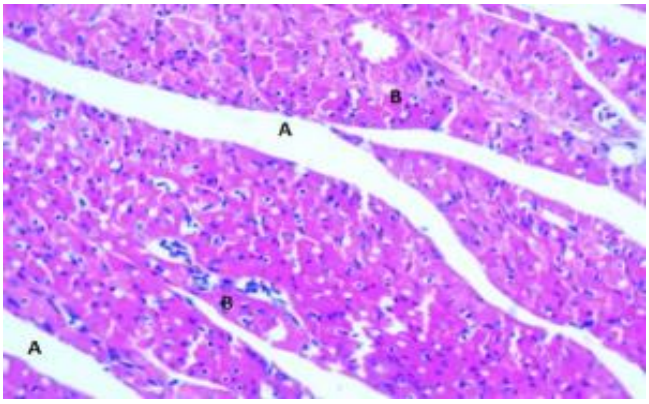

Figure 14: Histological section of heart in racing pigeon. (A) Myocardial edema, and (B) infiltration of the inflammatory cell. H&E. 100X.

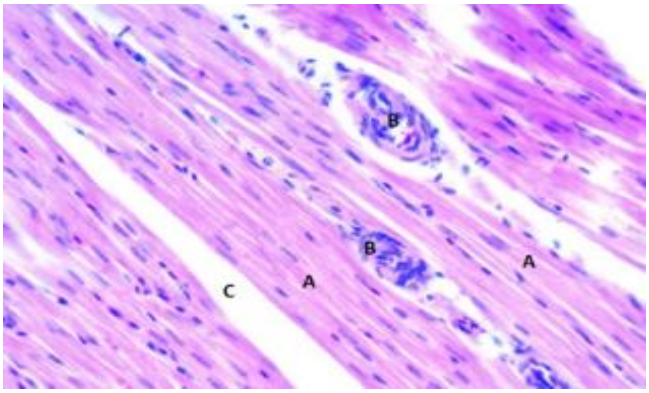

Figure 15: Histological section of the heart in racing pigeon. (A) Loss of striation of myocardial cells, (B) foci of lymphoplasmacytic cell infiltration, and (C) edema between myocardial cells. H&E. 400X.

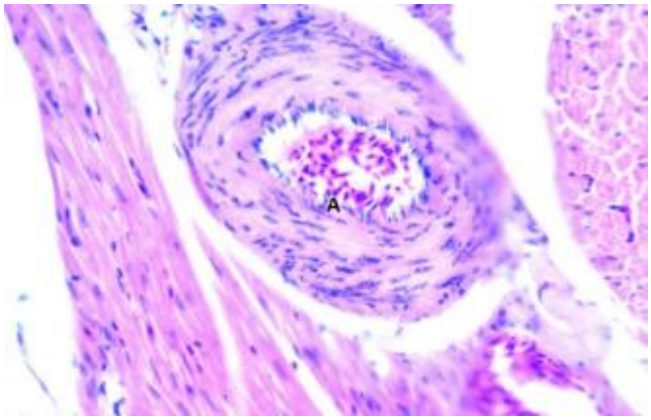

Figure 16: Histological section of the heart in racing pigeon. (A) Thickening of the wall of blood vessels (vacuities) with eosinophilic deposition. H&E. 400X.

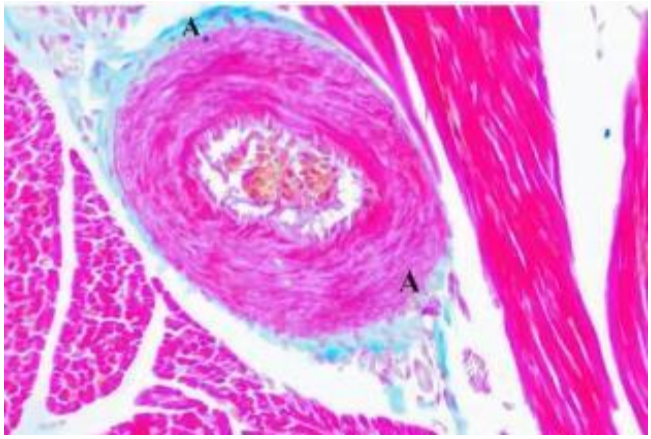

Figure 17: Histological section of the heart in the racing pigeon. (A) Fibrosis in the myocardium and around the artery. Masson's trichrome stain. 400X.

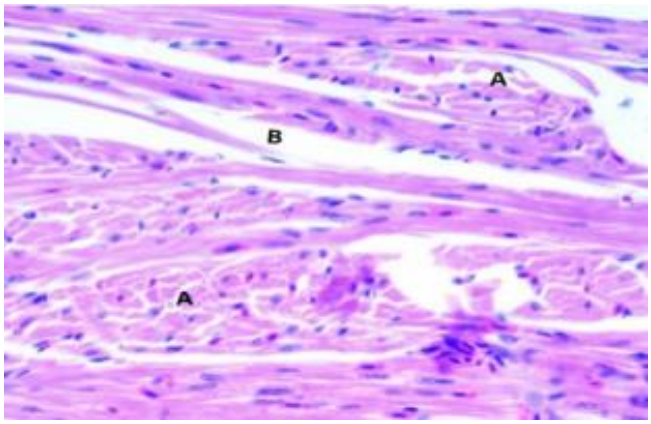

Figure 18: Histological section of heart in racing pigeon. (A) Myocardiopathy in the myocardium, and (B) edema between cardiac muscle fiber. H&E. 400X.

Discussion

Few reports are documented about histopathological changes in racing pigeons infected naturally with APMV-1; the intensity of histopathological lesions corresponds to a high level of virus expression. The brain, liver, kidney, and heart showed more persistent and notable histopathologic changes. Lymphoplasmacytic encephalitis with neuronal necrosis and degeneration was present in the brain lesions, meningeal blood vessels congestion, vasogenic edema, focal area of encephalomalacia, neuronal vacuolation associated with areas of Purkinje cell degeneration and necrosis. Lesions were severe enough to affect the normal development of birds and cause significant neurologic defects. These changes are agreed with the (19,27,28). The clinical signs and pathological lesions caused by the increased level of interleukin-6 (IL-6), which is an increase in pPMV-1 infections in all tissue infected, the IL-6 expression promotes tissue damage and inflammation in chickens and pigeons infected with PPMV-1 (27,29,30). Neuronal necrosis and degeneration occur due to a rich blood supply, which causes an increased rate of virus infection in the brain's tissue and local virus replication in this area (30-32). Liver histological sections show nuclear pyknosis, necrosis, and degeneration of hepatocytes. Other tissue sections showed necrotic foci with inflammatory cell infiltration, dilatation of sinusoid, and other tissue sections showed fibrosis around blood vessels with vacuities; the type and severity of lesions are related to many factors like virulence of the virus, immunity of the host, age of the bird and tropism of the tissue for virus, cytokines over production from virus infection lead to cytokine storm thus increase inflammation to the host, the distribution of fibrosis as a result of hepatocellular injury and chronic hepatocellular fibrosis it may be associated with steatohepatitis and primary cholangitis (19,24,33,34). Kidney sections revealed foci of lymphoplasmacytic infiltrations, cell swelling, and epithelial cell lining renal tubule degeneration. Some sections show hyaline cast and hemorrhage, virus disseminated by a different mechanism like viremia. Also, the lymphocyte can be infected with the virus; thus, the circulated infected lymphocyte shares virus dissemination, including kidneys (35-39). Significant lesions were evident in the heart, massive infiltrations of lymphoplasmacytic and myocarditis accompanied by myofibers necrosis, loss of myocardial cell striations, and thickening in the wall of blood vessels because NDV-infected pigeons cause alteration in electrocardiograms of the heart because of the heart is highly functional organs (39-43). To distinguish a scar area (collagen type one 1) from a healthy one, we used the Masson’s trichrome stain so millions in an ischemic heart, cardiomyocytes are destroyed, which causes fibrous scar tissue to develop (44-46).

Conclusion

In recent years, infection with PPMV-1 in pigeons has become more prevalent and causes a real threat to poultry since the pigeons are the vectors of the virus. Our study explains the necessary investigation of PPMV-1 infection in pigeons. Results revealed that while numerous tissues experience gross and histological abnormalities due to the PPMV-1 infection, the brain, liver, kidney, and heart are the most severely afflicted organs. The results showed that the virus replicates in any tissue organ and thus causes lesions and severe problems in chickens.

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Conflict of interest

The author has no financial or personal ties to groups or individuals who might have improperly influenced the paper's content.

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التحقيق في التغيير النسجي المرضي للفيروس المخاطاني الأول في حمام السباق المصاب طبيعيا

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الخلاصة

هدفت الدراسة الحالية إلى التحقق من إصابة حمام السباق الزاجل بعدوى الفيروسات المخاطانية، بناءً على العلامات السريرية والتغيرات النسيجية والكيميائية النسيجية، والتي تسبب زيادة في معدل الوفيات في قطعان الدواجن ويطلق عليها فيروس الحمام المخاطاني. شملت عينات الدراسة ٤٥ طائرا التي جمعت من مناطق مختلفة في مدينة الموصل حيث لوحظ عليهم علامات عصبية والتي تعتبر من العلامات المرضية الواصمة لعدوى الفيروسات المخاطانية مثل الرعشة والترنح وشلل الساق والجناح والتواء الرقبة. أجريت الدراسة على بعض الأعضاء الأكثر تأثراً بعدوى الفيروسات المخاطانية والتي تمثلت بالقلب والدماغ والكبد والكلى، عيانيا مثلت آفات الدماغ نسبة ٦٦,٦٪، آفات القلب نسبة ٣٧,٧٪، آفات الكبد نسبة ٥٥,٥٪ في حين كانت الآفات الكلوية بنسبة ٤٢,٢ ٪ على التوالي. أظهرت نتائج الفحص النسيجي لهذه الأعضاء العديد من التغيرات النسيجية المتمثلة في التغيرات التنكسية والنخرية والالتهابية في جميع الأنسجة المدروسة بالإضافة إلى وجود تغيرات في الأوعية الدموية مع تليف في بعض المقاطع، في حين أظهرت المقاطع النسجية المصبوغة بملون الماسون الثلاثي الكروم تفاعلا شديدًا لألياف الكولاجين في الكبد والقلب. نستنتج من هذه الدراسة الحالية وجود تغيرات نسيجية مرضية وكيميائية في أعضاء حمام السباق والمصاب طبيعيا بالفيروس المخاطاني -١و حيث يعتبر الحمام مصدرا خطيرا لنقل للفيروس والذي يتكاثر بدوره في جميع أعضاء الجسم ويشكل تهديدا خطيرا ومشكلة لقطعان الدواجن.